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Factors affecting sleep in the critically ill: An observational study☆,☆☆,★,★★

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ABSTRACT

Purpose: The aims of the current study were to describe the extrinsic and intrinsic factors affecting sleep in critically ill patients and to examine potential relationships with sleep quality.
Materials and Methods: Sleep was recorded using polysomnography (PSG) and self-reports collected in adult patients in intensive care. Sound and illuminance levels were recorded during sleep recording. Objective sleep quality was quantified using total sleep time divided by the number of sleep periods (PSG sleep period time ratio). A regression model was specified using the "PSG sleep period time ratio" as a dependent variable.
Results: Sleep was highly fragmented. Patients rated noise and light as the most sleep disruptive. Continuous equivalent sound levels were 56 dB (A). Median daytime illuminance level was 74 lux, and nighttime levels were 1 lux. The regression model explained 25% of the variance in sleep quality ($P = .027$); the presence of an artificial airway was the only statistically significant predictor in the model ($P = .007$).
Conclusions: The presence of an artificial airway during sleep monitoring was the only significant predictor in the regression model and may suggest that although potentially uncomfortable, an artificial airway may actually promote sleep. This requires further investigation.

1. Introduction

Patients treated in intensive care units (ICUs) frequently experience poor quality sleep [1,2]. The quantity of sleep may be acceptable, but it is highly fragmented; thus, stage 1 and 2 sleep is prolonged and slow wave and rapid eye movement (REM) sleep is short [1,2]. A multitude of extrinsic and intrinsic sleep disruptive factors, many of which are interrelated, may be responsible for sleep disruption in ICU patients [3]. Intrinsic factors are patient related and include prehospital sleep quality, the inflammatory response, pain, and circadian rhythm disruption.

Polysomnographic (PSG) sleep data and data for variables potentially associated with sleep disruption (eg, environmental sound and illuminance levels) were collected from 53 ICU patients [1]. In order to devise and test future interventions to improve sleep in ICU patients, we planned to analyze the data to explore the relative effect of these factors on sleep arousals, but because sleep was highly fragmented (median sleep period without waking, 3 minutes), other methods of analysis were necessary to explore factors that disrupt sleep in ICU patients. We sought to model sleep disruption in this cohort of ICU patients. Thus, the aims of the current study were to describe the extrinsic and intrinsic factors affecting sleep in critically ill patients and specifically to:

1. examine the relationships between the extrinsic and intrinsic factors affecting sleep and sleep quality, and
2. develop a regression model to explain the variance in sleep quality in ICU patients.

2. Methods

2.1. Participants and setting

Adult ICU patients older than 17 years with an anticipated ICU length of stay greater than 24 hours were invited to participate. Exclusion criteria were as follows: history or evidence of sleep disorder (eg, obstructive sleep apnea), history or evidence of psychiatric illness, known diagnosis of dementia, drug or alcohol withdrawal at the time of

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screening, and central neurologic impairment (eg, brain trauma confirmed by scan, hypoxic brain injury, suspected encephalopathy, seizure disorder, or drug overdose).

The study was conducted in a 36-bed general and cardiothoracic adult ICU at a 600-bed metropolitan hospital in Sydney, Australia. This hospital was a tertiary referral facility for specialty services such as cardiac, spinal, renal, neuroscience, and burns. Twenty-four hour sleep recording (PSG) took place in 5 of the 8 patient rooms in the ICU. Approval to conduct the study was provided by the Human Research Ethics Committees for the Hospital (protocol number: 0809-201M (SP)) and the University of Technology Sydney. Patients gave informed consent to participate which was confirmed by their closest proxy who also provided written informed consent. The sleep data collected from participants and some environmental sound and illuminance level data collected in this study have been previously published [1]. This article contains additional information about the data concerning sleep disruptive factors and their relationship(s) to sleep quality (ie, data were analyzed post hoc to explore potential sleep disrupting factors).

2.2. Instrumentation

2.2.1. Sleep measurement

Sleep was measured objectively using PSG for one 24-hour period (this protocol is described elsewhere [1]) and subjectively at the conclusion of PSG monitoring and in the hospital ward. Patients self-reported on the quality their sleep before hospitalization (Insomnia Severity Index [ISI] [4] and responded to a question in the Sleep in Intensive Care Questionnaire (SICQ) [5], in ICU (using the Richards Campbell Sleep Questionnaire (RCSQ [6]) and on the Hospital ward (RCSQ and SICQ)).

2.2.1.1. Insomnia Severity Index. The ISI was developed to identify clinically significant insomnia (based on *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* [7] diagnostic criteria for the condition). Concurrent validity ($r = 0.65$) was investigated in unpublished work by Morin and reported by Bastien et al [4]. Lower scores on this instrument indicate better sleep (a cutoff score of 15 is indicative of significant sleep difficulty).

2.2.1.2. Sleep in Intensive Care Questionnaire. The SICQ was administered to assess the patients' perception of sleep disturbances and their sleep in the ICU. The SICQ [5] contains 7 questions, some with more than 1 item. Responders are requested to rate their overall sleep quality at home and in the ICU (and at 3 different times during their stay) using the SICQ. In addition, ratings on daytime sleepiness are included, along with sources of perceived sleep disruption and noise. Items are rated on a scale of 1 to 10. Ten is the most desirable score for items contained in questions 1 to 5, and 1 is the most desirable score for items in questions 6 to 7. The SICQ was developed in the 1990s in North America to assess ICU patients' sleep quality and the factors that contributed to sleep disruption while they were in ICU [5]. There are no reports of formal validation or psychometric tests of the SICQ. However, the authors performed pilot testing on 43 patients and as a consequence added question 7 regarding noise disruption.

2.2.1.3. Richards Campbell Sleep Questionnaire. The RCSQ [6] comprises five 100-mm visual analog scales: sleep depth, latency, awakenings, time awake, and quality of sleep. Responses are scored by measuring the distance from the low end of the scale to the mark made by the patient. The total score for the RCSQ is calculated by adding the score for each visual analog scale and dividing by 5. High scores indicate good-quality sleep.

The RCSQ was pilot tested in a medical ICU (n = 9, 100% male, 14 nights) [8] and validated in a more extensive investigation involving 70 male patients [6]. The correlation between total RCSQ score and PSG sleep efficiency index was moderate ($r = 0.58$, $P < .001$); the total RCSQ score was able to predict 33% of the variance in the sleep efficiency index [6]. There are no published RCSQ data for healthy individuals on which to base a comparison or provide cutoff scores for poor, moderate, or good sleep.

2.2.2. Sound level measurement

A portable sound level meter and analyzer (Model 2250; meeting international standard IEC 61672-1), microphone (Model 4189) attached to a 3.0-m extension lead, and a calibrator (Model 4231; Brüel and Kjaer, Nærum, Denmark) were used. Sound level meter software BZ7222 ver 1.5, Frequency analysis software BZ7223 ver 1.5, and logging software BZ7224 ver 1.4.1 were used (Brüel and Kjaer). The sound level meter was programmed to record sound pressure broadband parameters along with LZ spectra at a sampling and logging frequency of 1 sample per second for 24 hours during PSG recording. Maximum input level was 141.07 dB, and 1/3 octave bandwidth was used for the sound spectra. The "Logging" mode was used. Calibration was performed prior to each study at 1000 Hz and 94 dB as a reference output. The microphone was placed approximately 1 m above the patient's head in order to record the sound level the patient was exposed to. The continuous equivalent sound level was measured in decibel (A). The LA_{eq} is the time averaged sound level using the "A" frequency weighting (which corresponds closely to the response of the human ear at low sound pressure levels). Technically, this is 10 times the logarithm to base 10 of the ratio of a root-mean-square sound pressure during a specified time interval. LA_{eq} is commonly used in occupational health assessments of sound pressure exposure. The greatest absolute sound levels were measured and reported as LC_{peak}. Technically, this is 10 times the logarithm to base 10 of the ratio of the greatest absolute instantaneous sound pressure level using the "C" weighting frequency filter (which is responsive to sound signals at higher sound pressure levels than 100 dB) during a specified period (1 second in our study).

2.2.3. Illuminance level measurement

The T-10 illuminance meter (Konica Minolta (c) , Marunouchi, Chiyoda, Japan) was used to record light levels throughout the 24-hour PSG data collection period. The T-10 is an illuminance meter used by light engineers. It was attached to the laptop computer via a serial port to USB port converter. Automatic calibration occurred when the meter was switched on. A 1-minute sampling and recording period was used. The illuminance meter sensor was placed on the pillow beside the patient's head in order to record the illuminance level the patient was exposed to as accurately as possible.

2.3. Data management and analysis

Clinical and demographic data were analyzed using SPSS (SPSS, Chicago, Illinois). Sound level reports were generated using the Utility software for Handheld Analyzers BZ 5503 (Brüel and Kjaer). Sound and illuminance level data were analyzed using Excel (Microsoft, Redmond, Washington). Summary statistics (median sound level for the entire recording, 2100-0600 and 0600-2100 hours) were calculated.

Normally distributed continuous data were described using means and SDs, and nonnormally distributed continuous data were described using medians and interquartile ranges (IQRs). Categorical data were described using frequencies and percentages. Correlation coefficients (Pearson [r] for normally distributed continuous data and Spearman [r_s] for nonnormally distributed and point biserial correlation [r_{pb}] for dichotomous data) were used to examine the relationships between environmental factors and sleep quality as measured by PSG and patient self-report (RCSQ).

In order to "quantify" PSG sleep quality, we used the total sleep time divided by the number of sleep periods (PSG sleep period time ratio). We chose this in order to accommodate for the lack of

independence of each sleep period (even small amounts of slow wave sleep suppress the propensity to sleep afterward) and the slight variation in recording times between patients. (A sleep period was defined as >30 seconds of waveform activity on the PSG which was clearly representative of sleep.) We also selected this in preference to using the arousal index because in most patients, sleep periods were short (a few minutes), and therefore, the arousal index was (artificially) low. We theorized that arousal index would not be a good indication of sleep quality. There were also insufficient amounts (numbers of patients who experienced) of slow wave and REM sleep to use these variables in multivariate analysis. Patient self-report of sleep quality was defined as the total score for the RCSQ, which was administered after the PSG equipment was removed in ICU.

A linear regression model was specified post hoc to assess the influence of intrinsic and extrinsic factors on sleep quality as measured by the PSG sleep period time ratio; the model included predictors most highly rated by patients as sleep disruptive (that is sound and illuminance levels). The outcome was PSG sleep period time ratio, and the predictors were as follows: number of sounds peaks greater than 100 dB (C), presence of an artificial airway during sleep monitoring, administration of benzodiazepine medications, and median daytime (0600-2100 hours) illuminance level during PSG sleep monitoring. We chose to use the number of sound peaks greater than 100 dB (C) instead of the actual value (continuous variable) in our model for several reasons. First, we sampled at 1-second intervals, so there was a possibility of the result being statistically significant and not clinically significant (a 1-dB change is not clinically significant). Second, although sound levels as low as 45 to 55 dB [9] can be sleep disruptive, levels between 65 and 72 dB greatly increase the frequency of arousals [10], and peaks greater than 80 dB have been highly associated with arousals in ICU patients, we found that there were thousands of peak sounds greater than 80 dB in each 24-hour period, with little variability between patients.

3. Results

3.1. Patient demographic characteristics and PSG summary statistics

The patients were predominately male, and the majority were admitted to ICU with a nonoperative diagnosis (see Table E1, Supplementary Material).

Results of the ISI indicate that few patients were troubled with significant symptoms of insomnia prior to hospitalization. However, sleep monitoring with PSG indicated that although the median total sleep time was not excessively low (0500 [IQR, 0252-0714] hours) when they were in the ICU, it was highly fragmented and the quality was poor (little slow wave and REM sleep). Self-reported sleep quality was significantly poorer in the ICU than prior to hospitalization (SICQ: 7.06 ± 2.52 vs 4.50 ± 2.14, $P \leq .05$). The patients also reported poor sleep on the RCSQ (see Table E2, Supplementary Material).

3.2. Extrinsic sleep disruptive factors

Patients rated noise and light as the most sleep disruptive in ICU (Table 1). Continuous equivalent (56.60 ± 2.16 dB [A]) and background sound levels exceeded the World Health Organization standards on sound levels in hospital (47.20 ± 3.41 dB [A]). Illuminance levels were appropriate at night (median, 1 lux) but too dim for normal circadian rhythm during the daytime (median, 74 lux) (Table 1). The minimum median lux level was 23.35 lux, and the maximum was 351.00 lux. (Sixteen patients were exposed to a median daytime lux level of ≥100 lux.).

Table 1
Potential sleep disturbing factors

Patient self-reports: SICQ item, scale 0-10 (n = 45), mean ± SD,	
Noise	5.70 ± 2.75
Light	5.15 ± 2.61
Nursing interventions	5.05 ± 2.44
Diagnostic testing	4.49 ± 2.67
Vital signs	4.25 ± 2.12
Blood samples	4.01 ± 2.20
Administration of medications	3.84 ± 2.12
Environmental sound and illuminance levels	
Sound (n = 49)	
Leq (dB [A]), mean ± SD	56.60 ± 2.16
LF (dB [A]), mean ± SD	47.20 ± 3.41
Lpeak (dB [C]), mean ± SD	107.33 ± 10.32
Sound peaks >100 dB (C) per recording, mean ± SD, n	13.20 ± 10.37
Illuminance level (n = 45)	
Illuminance level during daytime (lux), median [IQR]	74.20 [43.54-139.80]
Highest illuminance level during daytime (lux)	3230.00
Lowest illuminance level during daytime (lux)	0.06
Illuminance level during nighttime (lux), median [IQR]	1.7 [1.13-2.52]
Highest illuminance level during nighttime (lux)	285.00
Lowest illuminance level during nighttime (lux)	0.00

Leq, continuous equivalent sound level; LF, background sound level; Lpeak, peak sound pressure level; daytime, 0600-2100 hours; nighttime, 2100-060 hours.

3.3. Intrinsic sleep disruptive factors

There were a limited number of potentially sleep disruptive intrinsic factors that were clinically significant; for example, mean pain score was low at less than 2/10, and mean anxiety level was low. However, approximately half of the patients had an artificial airway in situ during sleep recording, and a third of the sample were administered benzodiazepine medication during sleep recording (see Table E3, Supplementary Material). Both of these intrinsic factors were included in the regression model.

3.4. Associations between sleep disruptive factors and quantitative and subjective sleep quality outcomes

The presence of an artificial airway during sleep monitoring was positively associated with the PSG sleep period time ratio ($r_{pb} = 0.40$, $P = .004$; i.e., the presence of an artificial airway was associated with less sleep disruption); all other potential sleep disruptive factors were poorly associated with the PSG sleep period time ratio (e.g., the administration of benzodiazepine medications: $r_{pb} = 0.10$, $P = .502$; the number of sound peaks >100 dB [C] during PSG recording: $r = -0.18$, $P = .228$; and median daytime (0600-2100 hours) illuminance level: $r_s = 0.02$, $P = .876$).

There were no strong associations between subjective sleep quality in ICU (total RCSQ score) and potential sleep disruptive factors. The correlation coefficients with the total RCSQ score were low; for example, for the number of sound peaks greater than 100 dB (C): $r = -0.19$, $P = .279$; administration of benzodiazepine

Table 2
Multivariate analysis: regression model of the influence of intrinsic and extrinsic factors on the PSG sleep period time ratio (all patients with complete data for variables in model, n = 43)

	Unstandardized coefficient	*P*
No. of sound peaks >100 dB (C)	−0.09	.366
Presence of an artificial airway during sleep monitoring	6.82	.007
Administration of benzodiazepine medications	1.07	.678
Median daytime (06.00-21.00 hours) illuminance level	0.02	.137

Dependent variable = PSG sleep period time ratio; $R^2 = 0.246$; $P = .027$.

medications: $r_{pb} = -0.21$, $P = .184$; and presence of an artificial airway: $r_{pb} = 0.13$, $P = .425$.

A multiple regression model was developed to assess the effects of each of these variables on sleep quality. The predictor variables used in the model were the number of sound peaks greater than 100 dB (C), the presence of an artificial airway, administration of benzodiazepine medications, and the median daytime (0600-2100 hours) illuminance level during PSG sleep recording; the dependent variable was PSG sleep period time ratio. The model explained 25% of the variance in sleep quality ($P = .027$); the presence of an artificial airway was the only statistically significant predictor in the model ($P = .007$; Table 2).

4. Discussion

There was evidence of considerable sleep disruption in this cohort of ICU patients. Both qualitative and quantitative measures of sleep indicated that sleep quality was poor, it was highly fragmented, there was little slow wave and REM sleep, and patients' self-reports indicated that sleep was poor. These findings are comparable to the results of studies over the past 3 decades examining sleep in ICU patients [2,11–13].

One of the aims of the current study was to examine the relationships between the extrinsic and intrinsic factors affecting sleep quality in order to elucidate the main source of sleep disruption. The patients rated noise and light levels as the most sleep disruptive on the SICQ, and sound pressure levels were higher than the World Health Organization standards for hospitals [14]. In addition, illuminance levels were not conducive to the encouragement of normal circadian rhythm; a median of 74 lux (16 patients in the sample were exposed to a median lux level of ≥100; the maximum was 351 lux) is not sufficiently bright during daytime hours. A daytime lux level of 100 or higher is considered sufficient to suppress melatonin secretion and encourages normal circadian rhythm in most individuals [15]. The use of mechanical ventilation is a known impediment to sleep in ICU patients [16]. Medications, especially benzodiazepines, are commonly used to induce sleep but are widely understood to suppress slow wave and REM sleep [17]. However, the only significant correlation between sleep quality and potentially sleep disruptive factor was the "presence of an artificial airway during sleep monitoring." This relationship was positive, indicating that sleep quality may be better in the presence of an artificial airway. This is surprising because although the patients in the current study did not specify the presence of an artificial airway as sleep disruptive, there are frequent reports by patients in the international literature of the discomfort associated with artificial airways particularly the endotracheal (ET) tube [18–20]. For example, in a study in which former ICU patients were interviewed while still in the hospital, 68% remembered the discomfort associated with the ET tube and one third of the patients remembered that ET associated discomfort interfered with their sleep [18].

The other aim of the study was to develop a regression model to explain the variance in sleep quality in ICU patients. A regression model was fitted; a quantitative measure of sleep quality (the ratio between the number of sleep periods without waking and the total sleep time) was the dependent variable; and the predictor variables were two intrinsic factors and factors that the patients had rated as most sleep disruptive that is, noise and light. Interestingly, we found that the presence of an artificial airway during sleep monitoring was the only variable with a large regression coefficient, which was statistically significant, but sleep was not found to be related to sedation level measured by the Vancouver Interaction and Calmness Scale ($r_s = -0.21$, $P = .13$) or receipt of benzodiazepine medication ($r_{pb} = 0.10$, $P = .502$). We are unsure whether this is an anomalous result that cannot be explained or there is some potential explanation that needs further exploration.

This study has several limitations, the primary being the sample size. Polysomnography is a challenging and labour-intensive technique when used to assess sleep in critically ill patients. Our study comprised one of the larger sample sizes, but there was still considerable variation in demographic and clinical characteristics between patients. Sleep was measured once during the ICU patients' stay in ICU (in 1 ICU). Serial (or continuous) measurements may have provided a more complete picture of sleep disruptive factors. Intrinsic and extrinsic factors vary greatly during the illness trajectory; for example, plasma inflammatory mediator levels may be higher, the use of mechanical ventilation is more frequent, and activity levels and (concomitant) sound levels tend to be higher during the acute phase. (The ability to measure PSG continuously throughout the patient's ICU stay was limited by the availability of human and equipment resources.) Absolutely precise synchronization of the environmental illuminance and sound level monitoring equipment with the PSG recorder would have provided the opportunity to explore the relationship of awakenings with changes in these parameters. However, the resources were not available to do this either.

It is likely for many reasons that our model was not well specified. There are many other factors in ICU likely to affect sleep (the model explained 25% of the variation in the sleep quality), and our sample size was small, although larger than many in other ICU sleep studies. It is also likely that there is a nonlinear relationship between sleep quality and quantity and the many factors affecting sleep in ICU. For example, the effect of mechanical ventilation and sedative medications on sleep may be associated during procedures or during the acute phase of illness but not at other times. This type of relationship would be difficult to explore for one 24-hour episode of sleep monitoring during the patients' treatment in ICU. It is possible that the sleep disruptive factors are interrelated, and therefore, associations are difficult to measure and assess. There is considerable difficulty measuring sleep in ICU patients and exploring factors affecting sleep quality in this population. Interrater reliability for sleep was moderate for sleep technicians [1] so this unlikely to be the whole explanation (ie, classifying sleep when the patient was just behaviourally "still"). Although the interrater reliability for the analysis of sleep data was moderate [1], there remains the possibility that some data were misinterpreted; electroencephalographic anomalies were present in our data, and there are known problems with conventional sleep scoring in this population [21].

There is a current comment from ICU sleep researchers concerning the definition of sleep (or restorative state) and the most appropriate method of measuring it in ICU patients [21,22]. Arguably, until this is clearly elucidated, it will be difficult to be fully confident of the significant causes (contributing factors) for sleep disruption in ICU patients. Although this important work has yet to be undertaken, clinicians may look to evidence from epidemiologic studies for suggestions on how the ICU environment may be made more conducive to rest and sleep, for example, the sleep disruptive effects of aeroplane noise [9]. In addition, the discipline of sleep medicine has much to offer such as findings from research into the effects of hypnotics and sedatives [17,23].

5. Conclusion

Our study reconfirms and extends the findings of researchers investigating sleep in ICU patients. We developed a regression model to explore the effect of intrinsic and extrinsic factors on sleep in ICU patients. The results support findings from previous publications exploring this phenomenon; that is, the assessment of sleep and factors affecting it is problematic in the ICU setting.

The specified model explained 25% of the variation in sleep quality (defined as the total sleep time divided by the number of sleep periods). Surprisingly, the presence of an artificial airway during sleep monitoring had the largest positive effect after controlling for the other variables. This may suggest that although often a source of discomfort, an artificial airway may actually promote sleep. However, this requires further investigation to elucidate a possible underlying mechanism.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.jcrc.2014.05.015.

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